

POST-MORTEM NOTES OF DR. J. H. STARLING'S CASE
OF HEART-BLOCK.

BY THOMAS LEWIS.

[*Reprinted from "Heart."*
Vol. IX, No. 4, December, 1922.]

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POST-MORTEM NOTES OF DR. J. H. STARLING'S CASE OF HEART-BLOCK.

By THOMAS LEWIS.*

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IN the last volume of this Journal, Dr. J. H. Starling reported a case of heart-block. When this patient first came under observation he displayed transient attacks of block. Usually the heart's rhythm was normal, but from time to time a series of syncopal attacks occurred, and records of these attacks, taken by Dr. Starling, showed sudden cessation of the ventricular beat, the auricle continuing to contract. These attacks were shown to be related to vagal activity, being provoked by the act of swallowing and being abolished temporarily by atropine. A series of attacks occurred for the last time, and these were recorded, on May the 30th, 1919. On June the 28th and July the 31st, 1919, on January the 8th, August the 12th and November the 11th, 1920, complete block was recorded, the ventricular rate varying between 38 and 48 beats per minute. The previous account of this patient brings the history of the case to the last date.

Dr. Starling now writes that the patient was seen in January and June, 1921, and that the condition of the heart's rhythm was then unchanged. He was free from symptoms. "The harvest in this year commenced early, and he was doing his full day's work. On August the 3rd, 1921, he went out as usual to work, having made no complaint of feeling in any way different, and was found dead in the harvest field the same afternoon. Dr. Owens, of Long Stratton, kindly notified me of this event, and with some difficulty I was permitted to remove the heart from the body, but no further examination was allowed."

Dr. Starling forwarded the heart to me for examination.

Appearance of the heart. The heart, when emptied and with the vessels cut short, weighs 540 grammes. The epicardium is thickened slightly in places. The right auricle is dilated, the right ventricle is dilated and hypertrophied. The edges of the pulmonary valves are thickened; the tricuspid valve is normal. The left auricle is a little dilated and its endocardium irregularly thickened. The left ventricle is dilated and hypertrophied equally with the right chamber. There is a slight degree

* Observations undertaken on behalf of the Medical Research Council.

of thickening at the edge of the mitral segments, and a few small yellowish deposits in the flaps of the valve; otherwise the valve is normal. The aortic valves are a good deal thickened at their edges by fibrous tissue and small masses of calcareous tissue. The right and left anterior cusps, from which the corresponding and dilated coronary arteries spring, are thrown into one, the original line of sub-division being visible as a ridge in the sinuses of Valsalva. Below the junction of the posterior segment with its neighbour, the conjoined cusp, a tumour projects into the cavity of the ventricle. The tumour is rigid and finely nodular, and from its colour and consistence evidently contains calcareous matter. The mass is triangular in shape, the point projecting upwards and being firmly fixed to what was originally the right anterior cusp (see Fig. 1). The surface of the mass is glistening except over the summits of the small nodules of calcareous tissue; here the surface is rougher, but the calcareous tissue is not exposed to the blood stream. Below and behind this tumour the endocardium is densely thickened over a small area. The calcareous mass lies on the course of the left division of the bundle, the visible root of which can be traced into it from below. This mass does not project through the septum into the right ventricle. A few small fibro-calcareous patches are seen on the right or posterior papillary muscle. The base of the aorta is slightly atheromatous, more especially in and around the sinuses of Valsalva. The coronary vessels and their branches are dilated and atheromatous throughout their courses.

Histology.

A block of tissue, including the coronary sinus and septal flap of the tricuspid valve, on the right side, the nodule of calcareous tissue and corresponding aortic cusps on the left side, was excised. The calcified tissue was decalcified after hardening in formalin, and the whole embedded in celloidin and cut in horizontal serial sections from above downwards.

The auriculo-nodal junction is normal. The auriculo-ventricular node, which consists of very many interlacing fibres, loosely embedded in fat, is normal. The first part of the bundle consists of densely packed fine parallel muscular fibres and is normal. The connective tissue surrounding the first part of the bundle is sparsely invaded by lymphocytes. The veins in the node and bordering the node are unusually capacious. The first part of the bundle runs horizontally, in its second half it bends downwards, and, as it bends, it becomes flattened from side to side, and is a little more fibrous than it should be; there are also small collections of lymphocytes in its substance. Shortly after the bend, a space develops between the bundle and the fibrous septum; this space is unlined and is crossed by irregular connective tissue strands. The bundle is now approaching the aortic valve cusps and lies more to the left than is usual, being, in fact, closer to the cavity of the left than to that of the right ventricle; the septum is here densely

thickened by fibrous tissue in which are large unfilled spaces. A little later the bundle is level with the calcareous node shown in Fig. 1. This node consists of a mass of calcareous material broken into rounded masses by fibrous septa, the whole being enclosed in a thick fibrous capsule. At the level of the attachment of the aortic cusps, this calcareous mass lies on a fibrous base, the mass itself being entirely outside the limits of the septal musculature; but when lower sections are examined it is seen to penetrate more and more into the septum until it comes to occupy an almost central position still distorting the endocardium of the left ventricle and approaching closely to that of the right ventricle. When the bundle reaches this central mass it divides, and here it is heavily damaged, the capsule of the nodule being in contact with the bundle division. The bundle division is invaded by many fibrous strands from this capsule, and, like the capsule, is densely infiltrated with lymphocytes. The left division of the bundle is traced in the capsule of the nodule, pursuing its course for a short distance on its endocardial surface, where it is speedily replaced by fibrous tissue and can no longer be followed. Remnants of the right division are traced along the posterior and upper surface of the nodule for a short distance; it is rapidly incorporated and lost in the fibrous capsule. The whole of this region of the septum is fibrous and infiltrated.

Interpretation. There is no material interference with A-V node or A-V bundle over the first half of its course. A calcareous deposit densely covered in and divided by fibrous strands has its seat in the septum just in front and below the bundle division. It forms a saddle over which the bundle divides; the division is in close contact with the capsule of this nodule, and shows evidence of a chronic inflammatory process by which it is heavily invaded; the two bundle branches have their brief course in this fibrous capsule, in which all trace of them is speedily lost, remnants of the left branch continuing for a little way in the fibrous tissue beneath the left septal endocardium. There appears therefore to be a complete breach in the conducting tissues, which, so it is judged, must have been destroyed for a period of at least many weeks or months. The calcareous nodule appears to have developed centrally and to have expanded towards the left ventricle, dragging the bundle across with it and distorting it.

COMMENT.

This case presents points of unusual pathological interest. Some few years ago I reported¹ a case in which attacks of a similar kind occurred, namely, attacks of sudden cessation of the ventricular beat, the auricular contractions continuing. As in the present case, during the intervals between attacks the man was well and the mechanism of the heart was quite normal. Eventually the first patient developed complete block and died of it.

In these features, therefore, the case resembles that of Dr. Starling; but his case differs from it in an important respect; whereas my own case failed to react to atropine, his case did react, and clear evidences of a relation between the attacks and the vagus nerve were produced. Nevertheless, despite this preliminary evidence of vagal involvement, the man developed complete block and died of it, presenting post-mortem a complete and old-standing lesion of the conducting tract. This illustration warns us that in cases of transient block, even though clear evidence is forthcoming that the vagus is immediately responsible for the attacks of heart-block, it is still unjustifiable to conclude that an abnormal vagal action is primarily or wholly responsible for the patient's symptoms. In the present case the most reasonable view appears to be that owing to disease of the junctional tissues, natural vagal impulses were from time to time associated with conspicuously exaggerated reactions.

The second point of interest is the lesion itself. Through the kindness of Dr. H. G. Butterfield I have had the opportunity of examining another case in which a very similar lesion existed. It consisted of a rounded nodule of fibro-calcareous tissue, about a centimetre in diameter and projecting into the left ventricle from the septum in precisely the same situation as the lower part of the nodule here described. The lesion in his patient was not, however, quite so simple as that of Dr. Starling's case. The calcareous node joined a calcareous bar which was continued backwards in the septum at a slightly lower level as far as the aortic flap of the mitral valve. Dr. Butterfield's specimen was taken from a case of complete and chronic heart-block, but his sections failed to show an actual breach in the continuity of the bundle, which was but little invaded by actual disease. I call to mind a report* of a third case in which the lesion was similar to that described in the present note and occupied an identical position, projection into the cavity of the left ventricle. It is suggested that lesions of this kind may be consequent upon disease of a particular branch of a septal artery, and that it might be wise, when a further opportunity presents itself, to study in such a specimen the vascular supply of the septum by the method of injection.

REFERENCES.

- ¹ LEWIS. "Lectures on the Heart," New York, 1915, 104.
- ² STARLING. *Heart*, 1921, VIII, 31

* A published report to which I am no longer able to find the reference.

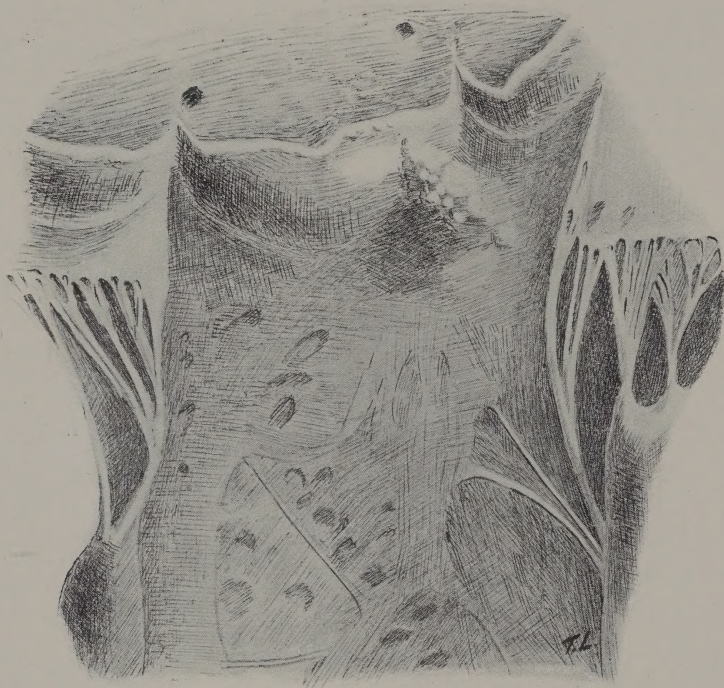


Fig. 1.

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